It appears that as a self-regulation strategy, meditation is equal to, but no more effective than, other self-control strategies such as biofeedback, hypnosis and progressive relaxation.

DEANE H. SHAPIRO, Jr, PhD

REFERENCES

Benson H: The Relaxation Response. New York, William Morrow, 1975

Otis L: Adverse effects of meditation, *In* Shapiro DH, Walsh RN (Eds): The Science of Meditation. New York, Aldine, 1981 (In press)

Shapiro DH: Meditation: Self-Regulation Strategy and Altered State of Consciousness. New York, Aldine, 1980

Shapiro DH: A clinical and physiological comparison of meditation with other self-control strategies: Biofeedback, hypnosis, progressive relaxation. Am J Psychiatry, 1981 (In press)

Shapiro DH Jr, Giber D: Meditation and psychotherapeutic effects—Self-regulation strategy and altered state of consciousness. Arch Gen Psychiatry 35:294-302, Mar 1978

Schwartz GE, Davidson RJ, Goleman DJ: Patterning of cognitive and somatic processes in the self-regulation of anxiety: Effects of meditation versus exercise. Psychosom Med 40:321-328, Jun 1978

The Diagnosis of Schizophrenia

THE ADOPTION BY THE American Psychiatric Association of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III, effective 1980) entails many changes in the diagnostic practices of American psychiatrists and other mental health professionals. An important inclusion in DSM-III is a particular set of diagnostic criteria for schizophrenia. These criteria render a clear verdict regarding a critical dispute that has lasted almost a century over opposing concepts of this most distressing and disabling of mental disorders.

Eugen Bleuler, who formulated the term schizophrenia, based his concept of the disorder on certain essential symptoms that could be identified in many persons who had never been psychotically ill. He believed that in mild grades and in its simple form, schizophrenia was extremely widespread in nonclinical populations:

> . . . the simple schizophrenics vegetate as day laborers, peddlers, even as servants. They are also vagabonds and hoboes. . . . On the higher levels of society, the most common type is the wife. . . , who is unbearable, constantly scolding, nagging, always making demands, but never recognizing duties.

Bleuler's broad concept of schizophrenia became rooted in American psychiatric diagnostic practice as exemplified by the very general diagnostic criteria established for schizophrenia in DSM-I (1952) and which persisted in DSM-II (1968). Many clinicians came to share the dismay of sociologically oriented observers that the label of schizophrenia, admittedly a sticky one, was being applied too often in a haphazard and potentially damaging way to patients (and nonpatients) with extraordinarily varied presentations in diverse settings and under diverse conditions of evaluation.

DSM-III stipulates that for a diagnosis of schizophrenia to be made, a patient must have had signs of the illness continuously for at least six months and that the six-month period must include an active phase with clearly psychotic symptoms (from an explicit list). Although DSM-III includes a list of potential prodromal or residual symptoms of schizophrenia, which encompasses Bleuler's essential symptoms, these are neither necessary nor sufficient for a diagnosis of schizophrenia.

The definitional requirement that schizophrenia be an illness characterized by psychotic symptoms and a continuous course of at least six months duration may help reduce the still existing skepticism that there is such a group of mental disorders and may improve treatment and research efforts for those who suffer from them. American psychiatry has taken a large step toward the Scandinavian practice of waiting five years from the onset of initial symptoms before confirming a diagnosis of schizophrenia.

MARVIN KARNO, MD

REFERENCES

Bleuler E: Dementia Praecox or the Group of Schizophenias. New York, International Universities Press, 1950

Diagnostic and Statistical Manual of Mental Disorders, 3rd Ed. Washington, DC, American Psychiatric Association, 1980

Tardive Dyskinesia

TARDIVE DYSKINESIA IS the most serious long-term side effect of all antipsychotic drugs. These drugs, in use for some 25 years, are the cornerstone of therapy in schizophrenia. Aftercare studies show substantial drug-placebo differences in relapse rates (favoring antipsychotic drugs) and, accordingly, it is accepted practice to prescribe maintenance antipsychotic drugs to most schizophrenic patients after their discharge.

Tardive dyskinesia consists of repetitive, involuntary movements that primarily involve, at least initially, the muscles of the face, lips and tongue. Involuntary mouthing, chewing, sucking, licking movements and tongue movements inside the mouth are frequent early manifestations. Later, the syndrome may include grotesque facial grimaces. choreoathetoid-type movements of the fingers, hands, arms and feet, abnormal trunk movements, peculiar gaits and abnormal diaphragmatic movements that result in grunting, difficult respiration and voice abnormalities.

Tardive dyskinesia occurs after several years (occasionally months) of antipsychotic drug therapy, with an estimated prevalence of between 15 percent and 50 percent after long-term therapy.

Patients are usually unaware of any abnormal

movements; thus, detection is entirely up to their physicians. If detected early, the dyskinesia will usually fade away if antipsychotic drugs are stopped; if drugs are continued, however, the dyskinesia is likely to become irreversible and may grow progressively worse.

Because there is no satisfactory treatment for tardive dyskinesia, we must try to prevent it. Much can be done by reducing drug exposure. Some, particularly those with misdiagnosed manic-depressive and panic disorders, are best managed with lithium carbonate and imipramine (Tofranil), respectively. Many cases of chronic schizophrenia can be maintained on much lower maintenance doses—doses as low as 1 mg of haloperidol (Haldol) or 50 mg of thioridazine (Mellaril) may suffice. Other patients, particularly those with families who can watch for the earliest signs of relapse, can be managed with intermittent drug treatment.

THEODORE VAN PUTTEN, MD

REFERENCES

Gardos G, Cole JO: Overview: Public health issues in tardive dyskinesia. Am J Psychiatry 137:776-781, Jul 1980

Klawans HL, Goetz CG, Perlik S: Tardive dyskinesia: Review and update. Am J Psychiatry 137:900-908, Aug 1980

Davis JM: Overview: Maintenance therapy in psychiatry: I. Schizophrenia. Am J Psychiatry 132:1237-1245, Dec 1975

Screening for Endogenous Depression: The Dexamethasone Suppression Test

A MAJOR GOAL of research in biological psychiatry has been to identify possible state and trait markers of psychiatric illness that might better predict drug response and prognosis than predictions based on clinical observation alone.

Although a variety of neuroendocrine and biochemical abnormalities have been reported for differing groups of psychiatric patients, most of these findings either have been difficult to replicate or have lacked clinical usefulness because of the complexity of the procedure involved, the expense of the test or the possibility of interference by psychotherapeutic medication. Also, in some instances, large statistical variances have made individual data equivocal. The first screening test to overcome these obstacles successfully has been the dexamethasone suppression test (DST), a test commonly used in the diagnosis of Cushing syndrome, but modified somewhat for usage with psychiatric patients. Current practice calls for the administration of 1 mg of dexamethasone at 11:30 PM and plasma samples assayed for cortisol at 8:00 AM on the day of administration and at 8:00 AM, 4:00 PM and 11:00 PM on the day after dexamethasone has been given. Approximately 50 percent to 60 percent of depressed patients show either a failure to suppress plasma cortisol levels with dexamethasone or an early escape from suppression (defined as 5 mg per dl or greater at any sampling point after administration of the drug). The principal usefulness of a positive finding at present is in assisting a clinician to make a differential diagnosis by helping to identify patients most likely to respond to antidepressant drugs. Drug-responsive depressions are thought to be more often endogenous as opposed to neurotic or characterological.

The DST also appears to help in the prediction of a beneficial response to lithium in catatonic patients and in the separation of primary from secondary forms of depression. Because the neuroendocrine abnormality appears to be state-specific (that is, patients usually show a normal response to dexamethasone with the resolution of their depression), serial DST's have been advocated as a possible indicator of how long to treat a patient with tricyclic agents.

VICTOR I. REUS, MD

REFERENCES

Brown WA, Johnston R, Mayfield D: The 24-hour dexamethasone suppression test in a clinical setting: Relationship to diagnosis, symptoms, and response to treatment. Am J Psychiatry 136:543-547, Apr 1979

Carroll BJ, Curtis GC, Mendels J: Neuroendocrine regulation in depression: I. Limbic system-adrenocortical dysfunction. Arch Gen Psychiatry 33:1039-1044, Sep 1976

Ettigi PG, Brown GM: Psychoneuroendocrinology of affective disorder: An overview. Am J Psychiatry 134:493-501, May 1977

Pseudodementia Syndrome

IN THE PSEUDODEMENTIA SYNDROME signs and symptoms that are consistent with irreversible organic brain disorders are predominant. However, in fact, these represent manifestations of nonorganic psychopathology, occurring typically as a major depressive episode. Failure to consider reversible causes of apparent dementing illness in the differential diagnosis of such patients can result in inappropriate treatment as well as an overdiagnosis of dementia. The pseudodementia syndrome often occurs in those age groups in which presenile and senile dementias are more prevalent, leading to further inaccuracies of diagnosis based on the notion that organic brain syndromes are a natural or expected concomitant of aging; however, this has not been shown to be the case.

The index of suspicion for pseudodementia is raised by several clinical features that help to differentiate the syndrome from true dementia. Among these are the following: progression of